

Clinical Experience with the Contraction Stress Test

H. A. SANDENBERGH, H. J. ODENDAAL

SUMMARY

During a period of 16 months, 1 170 contraction stress tests (CST) were performed on 767 women who were at high risk of losing their babies. The tests were positive in 42 patients, of whom 29 were subsequently delivered by caesarean section. Fetal distress, which necessitated caesarean section, occurred in 5 of 6 cases of intra-uterine growth retardation in which labour was induced. Abruptio placentae caused the intra-uterine death of 4 fetuses, 3 of which died within 7 days of a negative CST. The low perinatal mortality rate of 13 demonstrates the reliability of the CST in the evaluation of placental function in obstetric patients who are at high risk.

S. Afr. med. J., 51, 660 (1977).

Human placental lactogen and urinary oestriol determinations are used extensively to determine the metabolic and endocrine functions of the fetoplacental unit.^{1,2} However, several difficulties are encountered with the tests, especially in the collection of 24-hour urine specimens and in that results are not immediately available.

The ideal test should be reliable and easy to perform and interpret when serious doubt exists about the adequacy of placental function. Investigations have recently been directed to fetal heart rate patterns before the onset of labour as a means of determining fetoplacental respiratory function.^{3,4} Late deceleration patterns during contraction stress tests (CST) suggest placental respiratory insufficiency. The advantages of CST are the immediate availability of results and the relative ease of performance and interpretation.

PATIENTS AND METHODS

During a period of 16 months, 1 170 CSTs were performed on 767 patients. All these tests were performed by specially trained nurses. The CST was conducted with the patient in a 45° Fowler's position to reduce the occurrence of a supine hypotension syndrome. To exclude the latter, blood pressure was recorded every 10 minutes. Uterine contractions were recorded with an external labour transducer (HP 15136A) and fetal heart rate with an array ultrasound transducer (HP 15155A). These transducers were connected to a Hewlett-Packard cardiocograph (Model 8020A) and ultrasound amplifier (15180A). A recording speed of 2 cm/min was used throughout the test.

Department of Obstetrics and Gynaecology, University of Stellenbosch and Tygerberg Hospital, Parowvallei, CP

H. A. SANDENBERGH, M.B., CH.B.

H. J. ODENDAAL, F.C.O.G. (S.A.), M. MED. (O.&G.), M.R.C.O.G., M.D.

Date received: 20 October 1976.

Baseline uterine activity was recorded for a period of 10 minutes and was subsequently examined for contractions which lasted at least 45 seconds and occurred 3 or 4 times during a 10-minute period.

In the absence of sufficient contractions, oxytocin was administered intravenously, starting with an infusion rate of 1 mU/min. The infusion rate was doubled every 10 minutes until a maximum rate of 8 mU/min was obtained. When sufficient contractions failed to occur at this dose rate, the test was regarded as unsuccessful and was discontinued. Placenta praevia, premature rupture of the membranes, previous caesarean section and the danger of premature labour were regarded as contraindications for the administration of oxytocin. The test was interpreted as normal when no late decelerations were noted during contractions (Fig. 1). When repeated late decelerations occurred during uterine contractions, the test was regarded as positive (Fig. 2). The progress of all the tests was reported by one observer. When negative, the test was repeated after 7 days; for patients with diabetes or Rh-sensitization, the test was repeated sooner. When the test was positive the Bishop score of the cervix was assessed. When it was unfavourable for low amniotomy, a caesarean section was done. If favourable, the membranes were ruptured and a spiral scalp electrode and intra-uterine catheter were applied for internal monitoring of

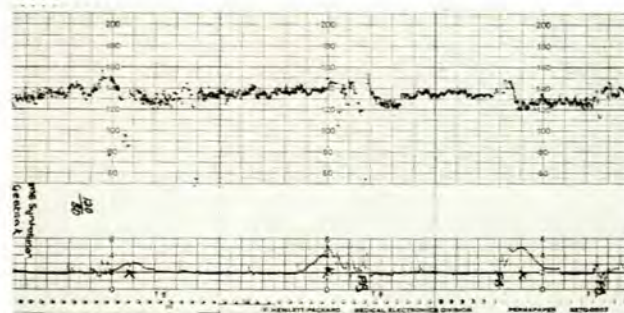


Fig. 1. Acceleration patterns are demonstrated during contractions.

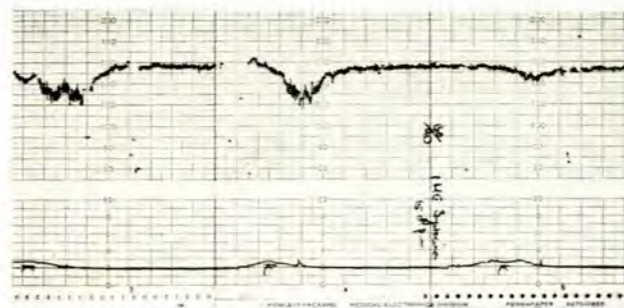


Fig. 2. Late decelerations are demonstrated.

the fetus during labour. When necessary, oxytocin was administered soon after the amniotomy.

The indications for the test are shown in Table I. Pre-eclampsia and post-term pregnancies were the most frequent indications for CST. This table also clearly demonstrates that all patients were classified as being at high risk. The large majority of patients had one or two tests.

TABLE I. INDICATIONS FOR CONTRACTION STRESS TEST

	%
Pre-eclampsia	30
Post-term pregnancy	29,4
Intra-uterine growth retardation	15,6
Poor weight gain	14,9
Diabetes mellitus	5,3
Poor obstetric history	3,9
Antepartum haemorrhage	0,7

RESULTS

More than 75% of the tests were interpreted as being normal. The tests were positive in 42 (3,6%) patients. In most of these the indications were pre-eclampsia or post-term pregnancy (Table II). Duration of pregnancy in the case of a positive test varied from 32 to 44 weeks (Fig. 3). No test was positive before the 32nd week of gestation. The results were uncertain in 1% of tests, 7,6% were unsuccessful and 8,3% could not be evaluated for other reasons.

Caesarean section was immediately performed in 17 patients because the cervix was unfavourable. Low amniotomy was done in 25 patients. In 9 of these, abnormal

TABLE II. CONDITIONS RESULTING IN A POSITIVE CST

Pre-eclampsia	17
Postmaturity	10
Intra-uterine growth retardation	8
Diabetes	3
Renal lesion	2
Hypertension	2
Other	3

TABLE III. METHODS OF DELIVERY

	Caesarean section	Normal	Forceps	Vacuum extraction	Total
Primary	17	0	0	0	17
Fetal distress	9	2	1	0	12
Other	3	7	1	2	13
Total	29	9	2	2	42

Fetal distress during labour: 48.

fetal heart rate patterns were noted in the early first stage of labour. These patients were delivered by caesarean section. Fetal distress occurred in 3 other patients, one of whom was delivered by forceps. Caesarean section was done for other reasons in a further 3 patients (Table III).

The date of the last normal menstrual period was known in only 34 patients in whom the CST was positive. In 12 of these the babies were small for gestational age according to the growth charts of Jaroszewicz *et al.*⁵ In 5 of these patients caesarean section was done as a primary procedure and in one it was performed for a cord prolapse after rupture of the membranes. Labour was induced in the remaining 7. In 6 of these, however, fetal distress occurred during labour, which necessitated abdominal delivery in 5 patients. The sixth patient was delivered by forceps. There was only 1 fetus with normal heart rate patterns.

Two neonatal deaths occurred after a positive CST had been recorded (Table IV). In the second patient extremely low urinary oestriol and serum human placental lactogen values were found several days before the stress test proved positive.

There were 5 neonatal deaths after negative CSTs. Oxytocin was administered to only 2 of these patients, and delivery followed 10 and 12 days after the test. In the remaining 3 patients, there were sufficient spontaneous uterine contractions (Table V).

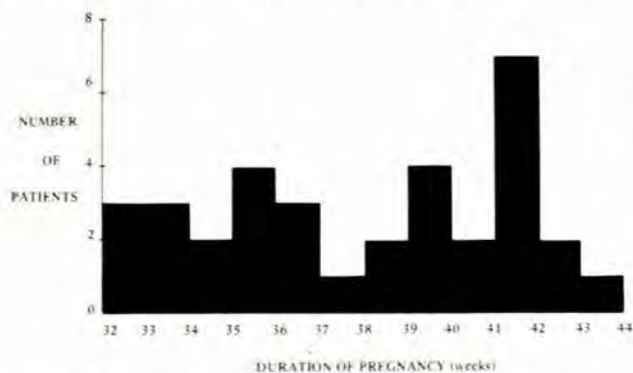


Fig. 3. Duration of pregnancy in patients with positive CST.

TABLE IV. NEONATAL DEATHS AFTER POSITIVE CST

Indication	Method of delivery	Apgar score	Mass (g)	Duration of pregnancy (weeks)	Cause	Age (days)
1. Pre-eclampsia	CS	10, 10, 10	1 290	35	Enterocolitis	10
2. Pre-eclampsia	(cord prolapse) CS	6, 8, 10	874	35	RDS	7

CS = caesarean section. RDS = respiratory distress syndrome

TABLE V. NEONATAL DEATHS AFTER NEGATIVE CST

Indication	Interpretation	Duration of pregnancy (weeks)	Test/delivery interval (days)	Oxytocin	Mass (g)	Cause	Age (days)
1. Poor history	Normal, with accelerations	39	4	No	2 912	Septicaemia	11
2. Uncertain dates	Normal, without accelerations	?	12	Yes	840 920	RDS	1
3. Pre-eclampsia	No contractions or accelerations	32	10	Yes	850	RDS	1
4. Poor weight gain	Normal, without accelerations	36	7	No	2 095	RDS	1
5. Uncertain dates	Tachycardia	36	1	No	2 410	RDS	1

RDS = respiratory distress syndrome.

TABLE VI. INTRA-UTERINE DEATHS AFTER NEGATIVE CST

Indication	Interpretation	Test/delivery interval (days)	Mass (g)	Duration of pregnancy (weeks)	Cause
1. Placenta praevia	Accelerations, no contractions	2	1 600	31	Rupture of membranes; cord prolapse
2. IUGR	Normal, without accelerations	2	2 200	39	Abruptio placentae
3. Pre-eclampsia	Normal, with accelerations	7	1 960	40	Abruptio placentae
4. Pre-eclampsia	Normal, without accelerations	5	1 510	36	Abruptio placentae
5. IUGR	Normal, without accelerations	10	1 400	40	Abruptio placentae

IUGR = intra-uterine growth retardation.

There were 5 intra-uterine deaths after a negative CST. One was due to a prolapse of the umbilical cord, while the others were due to abruptio placentae (Table VI).

DISCUSSION

In a selected group of patients at high risk (767), there were only 5 intra-uterine deaths. One was due to a cord prolapse after spontaneous rupture of the membranes in a patient who was hospitalized for placenta praevia. Since death in this case was due to acute fetal distress as a result of cord occlusion, it is highly unlikely that this disaster could have been predicted by a stress test. Abruptio placentae was the cause of intra-uterine death in the remaining 4 cases. The abruptio placentae occurred between 2 and 10 days after the test. It is therefore clear that a negative CST does not exclude the possibility of abruptio placentae. On the other hand, it is unlikely that the CST could have caused the abruptio placentae, since it occurred at the earliest 2 days after a CST. Furthermore, it is interesting to note that acceleration patterns were only noted in one of the tests where abruptio later caused intra-uterine death.

Oxytocin was administered in only 2 instances in which neonatal deaths followed a negative test. In the other 3, there were sufficient spontaneous uterine con-

tractions during the test. When oxytocin was administered, labour commenced 10 and 12 days after the test. It is therefore highly unlikely that stimulation of the uterus could have caused these preterm labours.

Two neonatal deaths occurred after positive tests. One of these, however, was caused by necrotizing enterocolitis which occurred 10 days after delivery. The other death could have been caused by the preterm delivery. Severe pre-existing placental insufficiency in this case, however, could easily have caused an intra-uterine death if the pregnancy had not been terminated.

Infants that were small for gestational age were born to nearly one third of the patients in whom the duration of pregnancy was known. Fetal distress developed in 5 instances in which induction of labour was attempted. In cases of intra-uterine growth retardation, placental function seems to be severely limited, and attempts to induce labour could cause further stress on the insufficient placenta. Abdominal delivery should be advised when it is known that the infant will be small for gestational age.

In several patients in whom labour was induced, abnormal fetal heart rate patterns were not observed. These could be regarded as suggestive of false positive tests. When these false positive tests were re-examined, it was noted that in 6 instances acceleration patterns, overstimulation, or a poor recording could have caused a

TABLE VII. SUMMARY OF CST REPORTED IN LITERATURE

	Incidence of IUGR	Incidence of caesarean section		Perinatal deaths	Incidence of positive tests	Incidence of false positive tests
		During labour	Total			
Cooper <i>et al.</i> ⁵	6/13	—	10/13	1/89	13/89	—
Ewing <i>et al.</i> ⁷	12/34	6/8	6/8	1/40	8/40	0/8
Farahani <i>et al.</i> ⁸	—	—	22/24	3/333	24/333	5/24
Freeman <i>et al.</i> ⁹	25/67	13/21	56/66	23/390	66/390	5/21
Gaziano <i>et al.</i> ¹⁰	3/7	0	7/7	0/72	7/72	—
Hayden <i>et al.</i> ¹¹	2/8	0	8/8	0/105	8/105	—
Ray <i>et al.</i> ¹²	5/15	—	12/15	4/66	15/66	1/15
Schiffrin <i>et al.</i> ¹³	2/9	1/1	4/7	3/120	9/120	3/9
Weingold <i>et al.</i> ¹⁴	—	—	—	5/154	14/154	6/14
Total	55/153 (36%)	20/30 (67%)	125/148 (84%)	40/1 369 29/1 000	164/1 369 (12%)	20/91 (22%)

wrong interpretation. However, in 4 instances, no reasonable cause could be found for the positive test. These could thus be regarded as truly false positive.

Experiences of other authors were also analysed (Table VII). It would, however, be incorrect to compare these results, since different definitions were employed for the relating problems. Freeman,⁹ who initiated most of the publications, did his original research as a blind study and this accounts for the higher perinatal death rate in his series. False positive tests were also differently interpreted. However, when the data are compared, the high incidence of growth retardation and fetal distress in cases where labour was induced is demonstrated. A low perinatal mortality is reported in the literature and is confirmed in this study. The incidence of false positive tests, however, was 8%. The figure reported in the literature is 22%. Clinical data as well as other placental function results and ultrasound growth curves should therefore also be considered when a clinical decision is made regarding the induction of labour or primary caesarean section.

Three intra-uterine deaths within 1 week of a negative stress test are reported in the literature.^{6,10} Two of these were due to congenital abnormalities and the other intra-partum death was caused by an umbilical cord entanglement. In this series, 4 intra-uterine deaths occurred within 1 week of a negative test. Rupture of the membrane in a

case of placenta praevia caused a cord prolapse in 1 patient. Abruptio placentae caused 4 intra-uterine deaths, of which 3 occurred within 7 days.

The low perinatal mortality in the high-risk population group of this series demonstrates the value of the CST. A negative test, however, fails to exclude the possibility of abruptio placentae.

We wish to thank the South African Medical Research Council for their support of this study.

REFERENCES

1. Spellacy, W. M., Buhl, W. C. and Birk, S. A. (1975): *Amer. J. Obstet. Gynec.*, **121**, 835.
2. Brown, J. B. (1974): *Clin. Perinatol.*, **1**, 273.
3. Freeman, R. K. (1975): *Amer. J. Obstet. Gynec.*, **121**, 481.
4. Freeman, R. K. and James, J. (1975): *Obstet. and Gynec.*, **46**, 255.
5. Jaroszewicz, A. M., Schumann, D. E. W. and Keet, M. P. (1975): *S. Afr. med. J.*, **49**, 568.
6. Cooper, J. M., Soffronoff, E. C. and Bolognese, R. J. (1975): *Obstet. and Gynec.*, **45**, 27.
7. Ewing, D. E., Farina, J. R. and Otterson, W. N. (1974): *Ibid.*, **43**, 563.
8. Farahani, G., Vasudeva, K., Petrie, R. and Fenton, A. N. (1976): *Ibid.*, **47**, 159.
9. Freeman, R., Goebelsman, U., Nochimson, D. and Cetrulo, C. (1976): *Ibid.*, **47**, 8.
10. Gaziano, E. P., Hill, D. L. and Freeman, D. W. (1975): *Amer. J. Obstet. Gynec.*, **121**, 947.
11. Hayden, B. L., Simpson, J. L., Ewing, D. E. and Otterson, W. N. (1975): *Obstet. and Gynec.*, **46**, 251.
12. Ray, M., Freeman, R., Pine, S. and Hesselgesser, R. (1972): *Amer. J. Obstet. Gynec.*, **114**, 1.
13. Schiffrin, B. S., Lapidus, M., Geeti, S. and Leviton, A. (1975): *Obstet. and Gynec.*, **45**, 433.
14. Weingold, A. B., De Jesus, T. P. S. and O'Keefe, J. (1975): *Amer. J. Obstet. Gynec.*, **123**, 466.